Understanding the Genesis of Trigger Point Pain

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Understanding the genesis of trigger points can point the way toward relief

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Myofascial trigger points have origins as deep as animal species bearing a musculoskeletal system. And while gravity makes it possible for life to be sustained on planet earth, it also becomes a foe for our muscles that work against it.

Our fascination with the myofascial system started with the original works of Travell & Simons and culminated in the 2001 publication of our book, The Manual of Trigger Point & Myofascial Therapy. Through the years, we’ve witnessed myofascial trigger point therapy become widely used by many health care providers for a variety of musculoskeletal problems.

Several studies demonstrate a high prevalence of pain due to myofascial trigger points. Fishbain et al. suggested that prevalence of pain originating from myofascial trigger points in a general pain practice can reach the level of 85 percent. The recognition of the myofascial pain phenomenon by the medical community as one of the key pain generators has led to the development and expansion of a variety of myofascial therapy interventions.

Teaching myofascial trigger point therapy for over 18 years to more than 30,000 therapists worldwide, we have come to realize that while therapists treat myofascial trigger points, many of them successfully, few of them have a deeper understanding of the pathophysiology of a myofascial trigger point.

The purpose of this article is to provide a summary of various mechanisms responsible for the genesis of myofascial trigger points. To do that, we must first discuss normal neuromuscular physiology and then identify those factors that can create myofascial trigger points when normal physiology fails.

‘Normal’ Physiology

In typical neuronal activity, the dendrite of an alpha motor neuron receives various excitatory and/or inhibitory stimuli. The sum of those stimuli will depolarize the axon hillock of the dendrite, creating a propagated potential traveling down the axon of the neuron via saltatory conduction jumping between nodes of Ranvier.

Upon arrival of the excitation potential at the presynaptic terminal, the action potential will briefly open voltage-sensitive calcium channels (VsCCs) located at the presynaptic terminal. Upon opening of VsCCs, calcium ions (Ca2+), abundant in the extracellular environment, cause a depolarization of the postsynaptic membrane and the creation of miniature end plate potentials (MEPPs). The voltage difference between the inside and outside cell membrane surfaces, along with the sum of the MEPPs, propagates the depolarization of the superficial cell membrane beyond the immediate area under the terminal bouton.

Any excess of neurotransmitter ACh that is not used is deactivated by the enzyme acetylcholinesterase (AChE). AChE breaks down ACh to acetone and choline. Choline gets reabsorbed by the presynaptic terminal to recycle and create new neurotransmitter, while acetone is being excreted from the body via urine, respiration and sweat.

It’s important to note that the entire muscle and each muscle fiber are surrounded by sarcoplasmic reticulum (SR), which is a tubular network that stores ionized calcium. As the depolarization travels down the T tubule, which is a component of the sarcoplasmic reticulum, it stimulates ryanodine receptors.

Activation of the ryanodine receptor releases Ca2+ from the sarcoplasmic reticulum into the cytoplasm of the muscle cell. The release of Ca2+ will trigger the interaction between actin and myosin filaments.

Also, it’s important to understand that a sarcomere is made up of myosin and actin...
filaments, which constitute the contractile component of the muscle. The actin filaments are covered by a large protein molecule called tropomyosin. Tropomyosin protects the actin heads from binding with the head of the myosin filament.

When calcium is released from the sarcoplasmic reticulum, it binds to troponin C. The binding of calcium to troponin C changes the shape of the tropomyosin molecule and uncovers the rest of the myosin-binding site of the actin filament. Myosin can now bind with the actin molecules, completing the power stroke of the myosin-actin binding.

The presence of ATP is necessary for the cycling and release of G-actin by the myosin head. The default position between the actin and myosin filaments is for the myosin head to be bound to the actin-binding site. The myosin head has also a nucleotide-binding site. The binding with a molecule of ATP causes a change in the configuration of the myosin head, which allows the release of actin from the actin-binding site.

Then, ATP hydrolyzes to ADP and a phosphate molecule. Remember that the last phosphate bond of ATP is a high-energy bond that creates enough energy to help the myosin head swing over and bind weakly to a new actin molecule. The crossbridge becomes now 90 degrees relative to the filaments.

The release of phosphate initiates the power stroke. In the power stroke, the myosin head rotates on its hinge, pushing the associated actin filament and passing it. At the end of the power stroke, the myosin head releases the ADP and resumes the tightly-bound rigor state. This completes the excitation/contraction coupling theory.

Two Proposed Theories
There are two proposed areas of dysfunction that can lead to formation of myofascial trigger points.

These are injury to the muscle with its sarcoplasmic reticulum, and the integrated hypothesis, occurring in the area of the neuromuscular junction.

Under the sarcoplasmic reticulum hypothesis, under conditions of overstretching, overshortening and/or overloading of the muscle, there may be injury to the muscle fibers and thus the creation of microtrauma or a macrotrauma. Along with the destruction of muscle fibers comes the destruction of part of the sarcoplasmic reticulum surrounding the injured area. When sarcoplasmic reticulum breaks, there is a release of ionized calcium, which will trigger a sustained muscle contraction.4

The second proposed theory, the integrated hypothesis, is one of the most prominent theories of myofascial trigger point genesis. It was introduced by Simons and supported by many others.6,7 This hypothesis involves three possible areas of dysfunction: the presynaptic area, synaptic cleft, and postsynaptic region.

Presynaptic area. As discussed earlier, upon opening of VsCCs, calcium ions abundant in the extracellular space enter the presynaptic terminal and cause presynaptic vesicles containing the neurotransmitter acetylcholine (ACh) to be released in the synaptic cleft.

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Liley has suggested that mechanical, thermal or chemical stimuli can increase the amounts of synaptic ACh demonstrated by increased spontaneous electrical activity (SEA) and identified by needle electromyography.6

This dysfunctional motor endplate hypothesis was also tested by Kuan and Hong, who observed a significant decrease of SEA when they injected myofascial trigger points with botulinum toxin type A, which blocks ACh release at the motor endplate. Mense et al. confirmed the hypothesis by injecting rat muscle with diisopropylfluorophosphate (DFP), a drug that increases release of ACh in the synaptic cleft.10 Sections of the muscle injected demonstrated significantly more contracted and torn muscle fibers compared to the part of the muscle not injected.

Furthermore, Chen and Grinnell showed that muscle stretch at the end plate by 1 percent can cause a 10 percent increase in ACh release.11 Kostopoulos demonstrated that SEA at a myofascial trigger point area may decrease with application of ischemic compression and passive stretch.12

Synaptic cleft. In the synaptic cleft, the enzyme acetylcholinesterase (AChE) deactivates any amount of neurotransmitter ACh that was not utilized. If AChE is deficient, ACh will continue to activate nAChRs, causing a continuous depolarization of the postsynaptic membrane. AChE is supported in the synaptic cleft by a structural protein (collagen Q) that anchors it to the plasma membrane.6 Drugs, chemicals, psychological and physical stress are risk factors for genetic mutations of collagen Q, and thus AChE deficiency.

Postsynaptic area. Postsynaptic nicotinic receptors (nAChRs) at the presence of neurotransmitter ACh become activated and open the sodium-potassium channel of the postsynaptic membrane. A mutational defect of the nAChRs may cause them to over-express, gain function and cause muscle hyperexcitability.

All the above cases cause a continuous partial depolarization of the postsynaptic membrane. At the same time, we mentioned that the presence of ATP is absolutely necessary to cause the binding and unbinding of myosin and actin filaments. But what if there is not enough ATP?

A partial depolarization of the postsynaptic membrane causes tightness in the muscle region with further tightening blood vessels in the immediate area, which causes a decrease of blood circulation. The reduced blood circulation creates a hypoxic state.

In a state of hypoxia, the cellular metabolism shifts to anaerobic cascade, providing only a fraction of the ATP produced aerobically. This produces an energy crisis in the vicinity of the muscle. ATP is important for three functions: the unbinding of actin-myosin filaments, directly reducing the release of synaptic ACh via a feedback mechanism, and as an energy source for Ca2+ reuptake from the sarcoplasmic reticulum. Lack of ATP produces a rigor mortis-like condition for the involved muscle region.

This overall state of ischemia, hypoxia and accumulation of metabolic waste produces the release of various nociceptive substances, such as by-products of cellular metabolism (potassium, hydrogen ions, oxygen free radicals), histamine from mast cells, serotonin from platelets, bradykinin from serum proteins, and prostaglandins, leukotrienes and substance P from injured muscle cells.

These products function as sensitizers of alpha and C-fibers transmitting pain stimuli, causing peripheral sensitization (decrease of the excitation threshold with firing with less activation), ectopic pain and even central sensitization along with the production of referred pain patterns. This process leads to muscle guarding and loss of flexibility, making the muscle vulnerable to a superimposed injury and in turn creating a vicious cycle.

Treatment Methods
There are various methods for the treatment of myofascial trigger points—some more invasive than others. Treatment mechanisms of action are beyond the scope of this article.

However, all treatments strive to normalize the pathophysiological cycle of the creation and sustenance of a myofascial trigger point. Treatment methods attempt to increase blood circulation, bring in more oxygen and ATP, and normalize the state of energy crisis. Treatment of the myofascially involved area must be precise and accurate, and target the reversal of the above-described pathophysiological mechanism.

References are available at www.advanceweb.com/pt under the Resources tab.

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